LETTER TO THE EDITOR

Role of mean platelet volume in acute exacerbation of chronic obstructive pulmonary disease

Letter to the Editor I have read the article published by Ulasli et al.[1] with great interest. The authors measured plasma mean platelet volume (MPV) in patients with chronic obstructive pulmonary disease (COPD) during acute exacerbation and during the stable period after 3 months of acute exacerbation. They compared the plasma levels of MPV between patients with COPD during the stable period and control subjects as well as between patients in the stable period and those with acute exacerbation. Mean MPV values during exacerbation were significantly lower than in patients during the stable period or in the control group (8.6 ±1.0 and 9.3 ±1.4 fl; both P <0.001). There was no statistically significant difference in MPV values between patients during the stable period and control subjects. The authors suggested that decreased MPV values might indicate increased systemic inflammation during exacerbation, and that MPV could be used as a negative acute phase reactant in acute exacerbation of COPD. This is a very interesting study, although we have a few critical comments concerning its methodology.

Firstly, in the methods section, the authors did not explain clearly the biochemical analysis. They did not mention the tube into which the blood sample was collected for the whole blood count. This is very important. Platelets exhibit a time-dependent swelling when blood samples are anticoagulated with ethylenediaminetetraacetic acid (EDTA); however, this swelling does not occur in the presence of citrate.[2] Also they did not mention the time interval between blood sampling and blood analysis. The recommended optimal measuring time of MPV is 120 minutes after venipuncture if the tube containing EDTA is used.[3] For a reliable MPV measurement, the potential effect of an anticoagulant on the MPV must be carefully controlled, either using an alternative anticoagulant (such as citrate) or standardizing the time delay between the sampling and the analysis (less than 2 hours). This is not clarified in the study.

Secondly, there are significant associations between MPV and diabetes mellitus, prediabetes, hypertension, hypercholesterolemia, obesity, metabolic syndrome, use of statins and selected antihypertensives, and atrial fibrillation.[4] These factors can greatly influence MPV values. The authors did not discuss these factors in patients with COPD and control subjects. Also, the factors should have been adjusted in both groups. Comparison of blood parameters alone can provide unreliable results.

The authors speculated that overproduction of proinflammatory cytokines and acute-phase reactants (interleukin 6 [IL-6], acute-phase proteins such as C-reactive protein, fibrinogen, and lipopolysaccharide-binding protein) can suppress platelet volume by interfering with megakaryopoiesis and the subsequent release of small-size platelets from the bone marrow. On the other hand, they differentiated IL-6 from other cytokines as an important proinflammatory cytokine that could induce thrombocytosis and affect platelet volume.[5] I think that during acute exacerbation of COPD, the effect of IL-6 can be negligible and the net effect of all proinflammatory cytokines and acute-phase reactants is the suppression of megakaryopoiesis and a decrease in MPV values.

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Author response We would like to thank the author of the letter for kind and valuable comments concerning our study.[1] We would like to highlight the points regarding the biochemical analysis and the study population.

Blood samples of the study population were collected in standard tubes containing EDTA and analysed within 1 hour after venipuncture. Therefore, we had standard EDTA tubes and time window for the analysis.[2,3]

In our study, healthy individuals without smoking history and systemic diseases were included as a control group, patients with COPD did not use any additional medications for other diseases, and the medications of patients and the characteristics of the control group had already been indicated in the manuscript.

Moreover, we did not evaluate the relationship between IL-6 and MPV in our study. In the study by Kaser et al.,[4] mentioned in the above letter, the pathway of thrombopoietin regulation by the inflammatory mediator, IL-6, in cancer patients, mice, and cell culture was demonstrated, indicating that the number of megakaryocytes or platelets by themselves might not be the sole determinant of megakaryopoiesis.[4] Further studies with a larger group of COPD patients are needed to explore the relationships between IL-6, other inflammatory cytokines, and acute-phase reactants with MPV in acute exacerbation period.

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